SPECTRAL PHONOCARDIOGRAPHIC STUDIES IN CONGENITAL HEART DISEASE

BY

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The principles and technique of spectral phonocardiography have been described in detail in earlier publications from this laboratory (McKusick *et al.*, 1954). The method provides detailed frequency-time-intensity plots of cardiovascular sound. It resembles in some respects the manner of operation of the auditory mechanism on these sounds. In essence the spectral phonocardiogram is made up of a large number of horizontal lines, each representing the output of an individual pass-band filter. In other words, in making the spectral phonocardiogram a selected segment of sound (in electrical form) is in effect put through a battery of a very large number of individual electrical filters each of which is tuned to a specific and different, although overlapping, pass-band.

Spectral phonocardiography has the following advantages over conventional oscillographic phonocardiography (McKusick *et al.*, 1955): (1) resolution in the time dimension is improved; (2) the wide intensity range of cardiovascular sound is better encompassed with simultaneous display of faint and loud components in their true proportions; (3) quality (timbre) is represented accurately; and (4) vibrations of non-cardiovascular origin are more easily identified. Spectral phonocardiography can do, or at least can be made to do, all the ear can. It can exceed the performance of the ear because (1) resolution in the time dimension is better, (2) it is not wed to one particular frequency response curve, (3) it is not handicapped by psycho-acoustic phenomena such as masking, and (4) permanent, quantifiable records are produced.

METHODS

Simultaneous recordings of heart sounds, electrocardiogram, and respiratory phase tracing were made on magnetic tape. The electrocardiogram and respiratory mark were recorded by means of frequency modulated carriers. A microphone (Altec 21–BR–150) of the condenser type was employed for pickup and transducing purposes. As a rule, quiet respiration was permitted during the recordings which were routinely made at the aortic area, at the pulmonary area, at the lower left sternal border (4th interspace), and at the apex. The tape recordings, raw data for the studies, were audited and selected sections analysed on the modified Bell sound spectrograph. In the spectral phonocardiogram the *time* dimension is on the *abscissa* as in most physiological recordings. The *ordinate* is *frequency* spectrum, not intensity. *Intensity* is represented by the *degree of blackness* of any given portion of the recording.

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The subject material consisted of approximately 100 patients with many varieties of congenital heart disease admitted principally to the Surgical Service of The Johns Hopkins Hospital for cardiac catheterization and/or surgical intervention. Atrial septal defect and pulmonary stenosis are the largest single diagnostic categories studied. Discussed here are the findings in these cases and in primary pulmonary hypertension, thoracic arteriovenous fistula, and congenital mitral stenosis.

Pure Pulmonary Stenosis, Valvular Type

The systolic murmur in these cases has had a Christmas tree appearance as in the case of the systolic murmur of aortic stenosis. Usually the frequency and intensity peak has been later in systole than in the murmur of aortic stenosis. In fact, its peak is at times so late in systole that the murmur has almost a crescendo construction (Fig. 1).

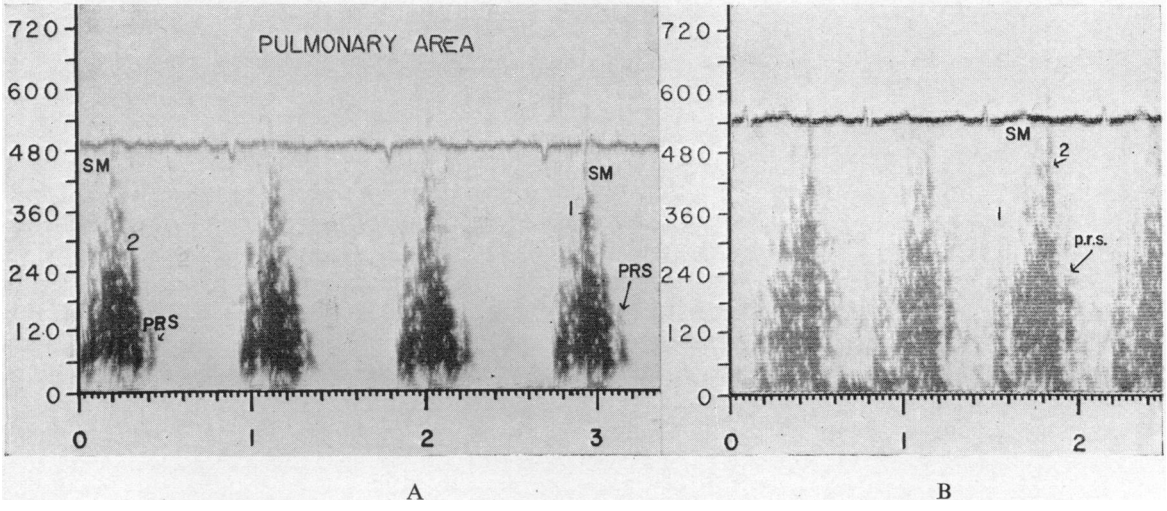


FIG. 1.—(A) Valvular pulmonary stenosis. Demonstrated are (1) the slightly split S_1 with the second component (tricuspid closure) greater; (2) the " Christmas tree " systolic murmur; (3) the unitary second sound; (4) the pulmonary reversal snap.
(B) In this instance the systolic murmur has its peak later in systole. Note the pulmonary reversal snap.

A second feature is the unitary character of the second sound which is loudest in the aortic area and results rather clearly from aortic closure alone.

Thirdly, loudest in the pulmonary area, a diminutive snap-like sound occurs 0·06–0·07 sec. after the aortic closure sound. It appears that this snap is a pulmonary closure sound. Since the pulmonary valve is reduced, in this situation, to a mere membrane with a central perforation, closure in the usual sense is inconceivable. It is more likely that the sound is produced when the membrane, domed into the pulmonary artery during ventricular systole, snaps in the opposite direction with fall in ventricular pressure. The mechanism is analogous to that conceived for the mitral opening snap. Because of the high systolic pressure in the right ventricle and the low pressure in the pulmonary artery the delay in this pulmonary reversal snap (as compared with a normal pulmonary closure sound) is readily understood. Angiocardiographic evidence for the proposed basis of the sound called here " pulmonary reversal snap " has been described to us by Johnson of Stockholm.

By spectral phonocardiography a pulmonary reversal snap of at least faint intensity is demonstrable in most cases of valvular pulmonary stenosis. Because of its low intensity in many cases and the masking effect of the preceding loud systolic murmur the ear is less successful in detecting the sound. Barritt (1954) found a " split second sound " in 11 of 33 cases. Abraham and Wood (1951) described a split pulmonary second sound in 12 of 19 cases of mild stenosis but

in all severe and moderate cases P2 was unitary and of presumed aortic origin. Diagnostically it can be as useful as the opening snap of mitral valve disease. The pulmonary reversal snap disappears after successful valvotomy.

In the fourth place, as was perhaps first indicated by Petit (1902), there may be an early systolic snap which is located shortly after the first heart sound and which may initiate the systolic murmur. This sound presumably results from the abrupt upward doming of the valvular diaphragm.

The low pulmonary diastolic pressure and small size of the orifice in the valvular membrane probably account for the rarity of a pulmonary diastolic murmur in these cases. More puzzling is the reason for the fortunate failure of Brock valvotomy to produce a diastolic murmur in the great majority of cases.

An atrial gallop was demonstrated at the left lower sternal border and/or apex in the majority of cases.

Recently, Vogelpoel and Shrire (1955) have pointed out that the character of the heart sounds permits one to differentiate pulmonary stenosis with intact interventricular septum and interatrial defect (with right-to-left shunt) from tetralogy of Fallot. In the latter condition the systolic murmur usually stops before or just with the aortic closure sound; in the former situation, the systolic murmur tends to extend through the aortic closure sound. Kjellberg *et al.* (1955) reported that in their experience the splitting of the second sound is the same in valvular and infundibular stenosis. However, in the latter type the murmur stopped abruptly with the aortic closure sound, whereas with valvular stenosis the murmur tended to extend beyond this point to the pulmonary closure sound.

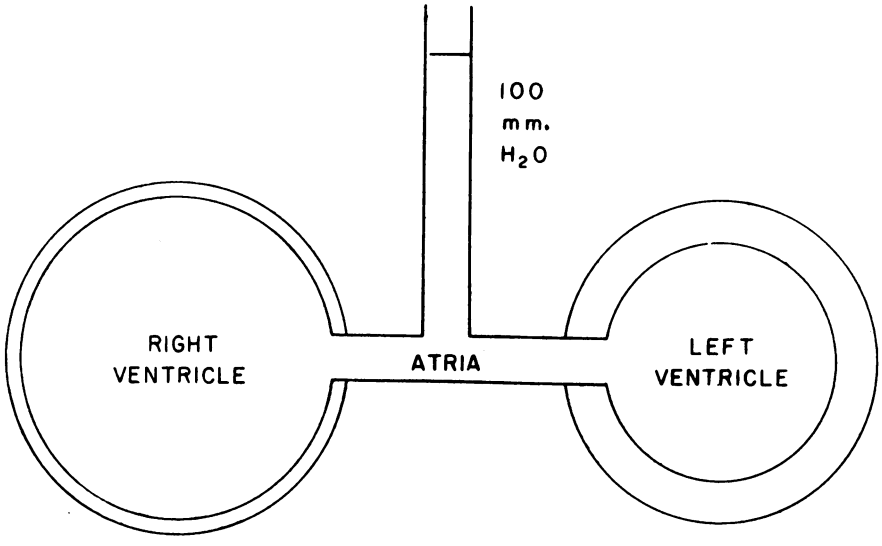
Atrial Septal Defect

For purposes of this discussion the auscultatory phenomena associated with this particular type of malformation can be classified in this way.

1. The systolic murmur(s).
2. Systolic click. (a) Early pulmonary systolic click. (b) Mesosystolic click.
3. The diastolic murmurs. (a) Apical rumble. (b) Pulmonary diastolic.
4. Splitting of the heart sounds.
5. Auscultatory features mimicking mitral stenosis.

Basic to a consideration of the systolic murmurs of atrial defect is the mechanism of the left-to-right shunt. Much has been written and speculated on this subject (Brannon *et al.*, 1945; Hickam, 1949; Uhley, 1942; Hall, 1949). Although several of the theories may describe factors contributing to the shunt, it seems most likely—as proposed by Dexter, Dow, and Maloney—that the shunt is *mainly* related to the difference in pressure-volume characteristics of the two ventricles. This is represented schematically in Fig. 2. Pressure-wise the two atria with a large septal defect are essentially one and the two ventricles are filled in diastole from the same head of pressure. The more distensible right ventricle, like the thinner walled balloon of the schema, will accept a larger volume than its thicker walled counterpart. This consideration does not exclude the possibility that some shunt may occur during ventricular systole as a result of the greater distensibility of the right atrium as compared to the left.

From this schema in Fig. 2 it follows that the bulk of the shunt occurs during diastole, not systole. The systolic murmur cannot, therefore, be considered to have its origin at the area of the defect. Satisfactory basis for the systolic murmur is provided by the anatomical state of the outflow tract from the right ventricle and by the increased volume of blood flowing through it. Relative pulmonic stenosis is to be expected from dilatation of the infundibulum below the pulmonary ring and of the pulmonary artery beyond. The existence of such stenosis is supported by demonstration of differences in systolic pressure across the pulmonary valve by catheterization in cases of atrial septal defect.



PROBABLE MECHANISM OF SHUNT IN I.A. DEFECT

FIG. 2.—The difference in the pressure-volume characteristics of the two ventricles is represented by balloons with different thicknesses of wall. Both are connected to the same pressure head by a T-tube. Obviously the thin-walled balloon dilates more.

The variable intensity of the systolic murmur, including its not infrequent absence, is in keeping with this rather indirect mode of genesis. Systolic murmurs may also result from relative tricuspid insufficiency and, in the case of low atrial septal defects of the ostium primum type with anomaly of the tricuspid and/or mitral valves, from regurgitation at these orifices, especially the latter. The association of a loud apical systolic murmur transmitted well to the axilla with other auscultatory signs consistent with atrial septal defect suggests that the defect is of the ostium primum type as does also the presence of left axis deviation by electrocardiogram.

The early systolic click (Fig. 3 and 4), usually loudest in the pulmonary area, is a frequent finding (Leatham and Vogelpoel, 1954). Dilatation of the pulmonary artery appears to be a *sine qua non* for its presence, but, in our experience, pulmonary hypertension seems to exaggerate this click. A snapping of the artery wall with the onset of ejection seems a plausible explanation.

A mid-systolic click is less commonly heard at the lower left sternal border (Fig. 5). It may be related to right ventricular hypertrophy and dilatation and may actually be produced by movement in costochondral or chondrosternal joints.

Apart from the apical diastolic murmur resulting from associated mitral stenosis, two types of diastolic murmurs are seen: (1) an apical diastolic rumble (Nadas and Alimurung, 1952), and (2) a pulmonary diastolic murmur due to dilatation of the pulmonary ring with or without pulmonary hypertension (Fig. 3 and 4). The apical rumble has been previously demonstrated by spectral phonocardiograph (McKusick *et al.*, 1954 *a*). The possibility that it has its origin at the shunt area must first be considered. Many of the defects have a remnant of fenestrated membrane covering them in part. (See Figs. V-2 and 11–44 by Gould.) The margin of what remains of the interatrial septum might be responsible for such a murmur in other instances. The fact that it is well heard at the cardiac apex is against this view, however, and in general high flow through the tricuspid orifice seems the most likely basis for this murmur.

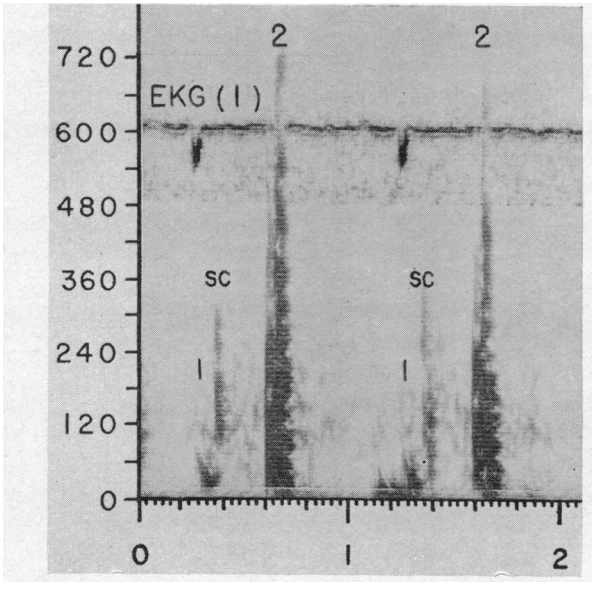


FIG. 3.—Pulmonary area with atrial septal defect. The first heart sound is virtually absent. The conspicuous early systolic click was confused for an unusually snappy S_1. S_2 is split; its second component (pulmonary closure) is greatly accentuated. The early systolic click initiates a soft systolic murmur. A faint early diastolic murmur follows the accentuated pulmonary closure sound.

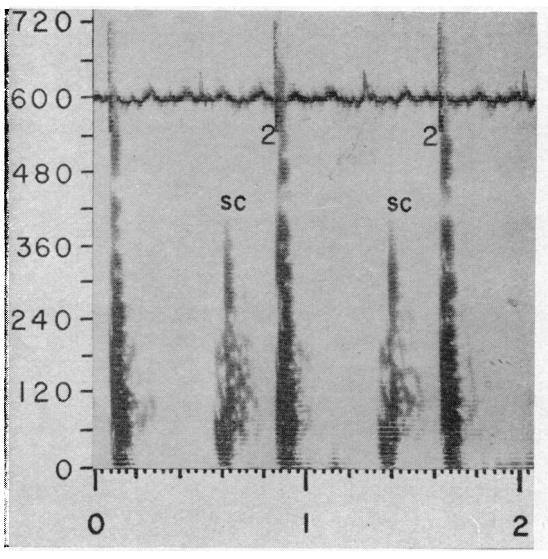


FIG. 4.—Pulmonary area in atrial septal defect. There is a conspicuous early systolic click initiating a faint systolic murmur. An even fainter diastolic murmur follows the accentuated S_2.

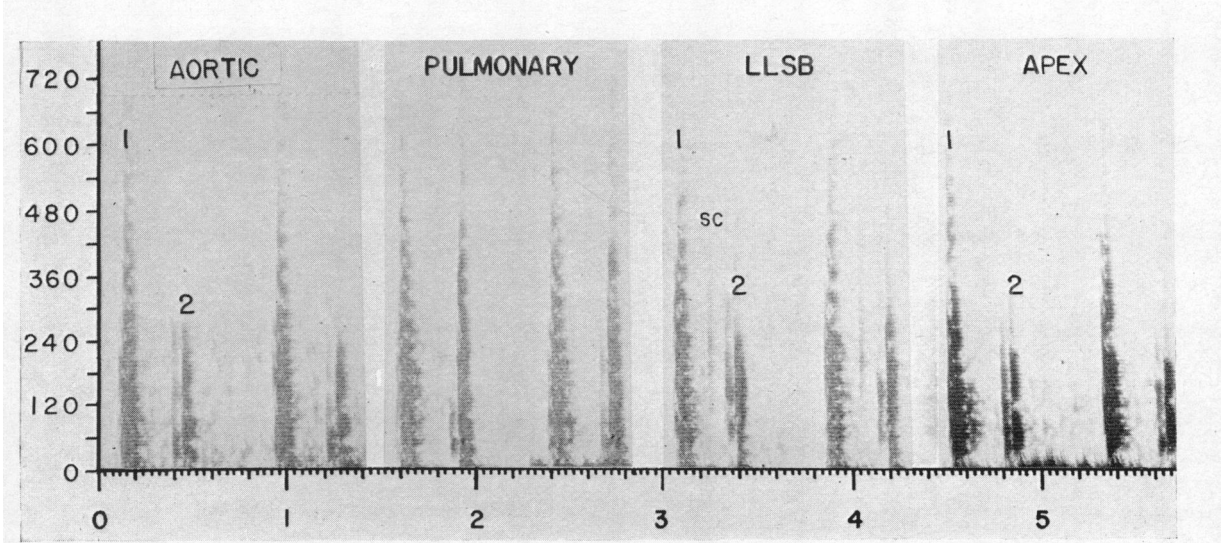


FIG. 5.—Atrial septal defect. Split S_2 with second component (pulmonary closure) accentuated. Mid-systolic click at LLSB.

The experience with other conditions, such as anæmia, mitral regurgitation, ventricular septal defect, patent ductus arteriosus etc. (see below), indicates that when dilatation of the ventricle and increased flow across the mitral orifice co-exist the stage is perfectly set for the development of turbulence and the corresponding rumbling or blubbering diastolic murmur. Both conditions —dilated right ventricle and torrential tricuspid flow—are satisfied in the cases of atrial communication. The audibility of the rumble in the "mitral area" is not surprising in the light of both the dilatation of the right ventricle and the clockwise rotation of the heart as a whole. The quality and timing of the murmur are identical with those of the murmur that results from high mitral flow on bases such as those listed above. Blount *et al.* (1950) described the disappearance of a mid-diastolic murmur at the fourth left interspace after complete surgical closure of the defect.

A pulmonary diastolic murmur occurred in two of eleven thoroughly studied patients. In one the murmur was accentuated during inspiration.

Ventricular asynchronism of pathological degree and resultant splitting of heart sounds occurs principally in two circumstances (Leatham, 1954)—delay of activation of one ventricle as in bundle-branch block, and discrepancy in the stroke volume of one ventricle as compared with the other. Both mechanisms are operative in some cases of atrial defect and the splitting of both heart sounds is exaggerated in these instances. The majority of cases, however, have splitting (Barber *et al.*, 1950) of the second sound (Fig. 3, 5, and 7) which is seemingly related to the great increase in stroke volume of the right ventricle over that of the left. In some cases, even without complete bundle-branch block, splitting of the first sound is also present. Prolongation of isometric contraction in the right ventricle cannot explain such splitting since by definition both the A-V and the arterial valves are closed during this period of the cardiac cycle. However, delay in tricuspid closure as compared with mitral closure is likely to occur in the manner described below, a process which is again related, at least indirectly, to the pressure-volume characteristics of the two ventricles. At the time the ventricles contract with closure of the A-V valves, filling in the case of the left ventricle is likely to be more nearly complete and the mitral leaflets will have floated up toward each other with partial closure. The dilated right ventricle, on the other hand, is likely to be still filling and the tricuspid leaflets to be widely separated. Not only will tricuspid closure be delayed slightly relative to mitral closure but its closure sound will be accentuated since the valves come together from a greater distance. In many ways this delay in tricuspid closure is comparable to

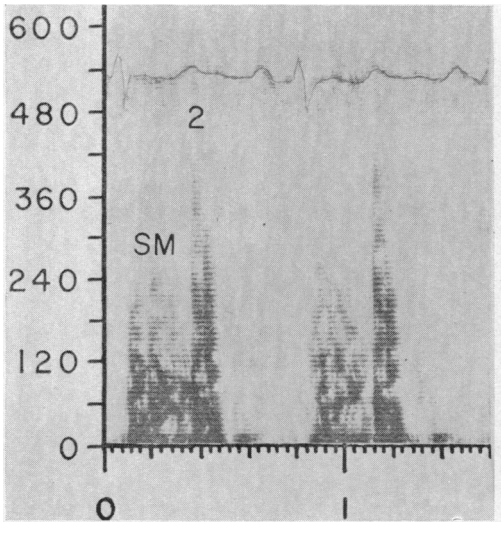


FIG. 6.—Split S_2 at LLSB in atrial septal defect.

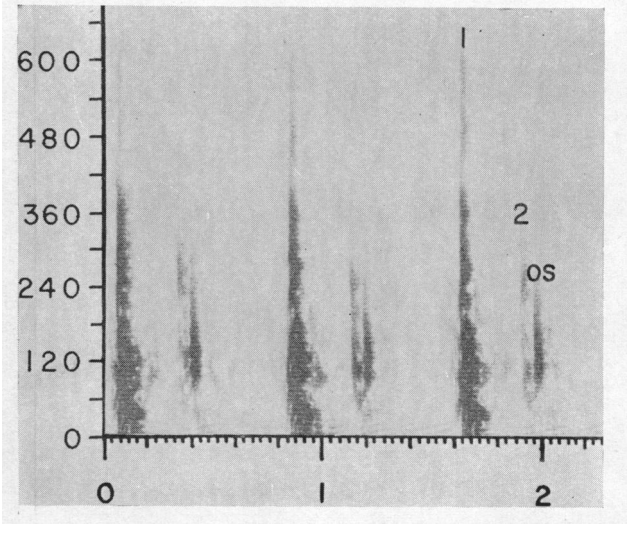


FIG. 7.—For comparison with Fig. 6: S_2 followed by mitral opening snap.

the QRS–S_1 delay, which occurs in atrial fibrillation with shorter diastolic periods as compared to longer ones (Luisada, 1941). Blount *et al.* (1950) illustrated the disappearance of splitting of S_2 after successful complete surgical closure of the defect.

Many of the auscultatory signs of atrial septal defect (as well as the radiological signs) suggest mitral stenosis and lead to that as the primary diagnosis or create an exaggerated impression of the incidence of Lutembacher's syndrome. These mimicking auscultatory signs are

1. the apical mid-diastolic rumble (see above),
2. the pulmonary diastolic murmur suggesting the Graham Steell murmur (Fig. 4),
3. the pulmonary early systolic click,
4. the split second sound which can be mistaken for second sound proper plus mitral opening snap, and
5. a crescendo construction of the first heart sound which simulates a presystolic crescendo and snapping first sound.

In the spectral phonocardiogram and to the experienced ear the mitral opening snap is usually distinguishable from a split second sound. In the case of a split second sound each component has the frequency pattern characteristic of valve sounds (Fig. 6); the opening snap, on the other hand, looks like a snap in the spectrogram (Fig. 7), has more homogeneous frequency content, and usually does not have its frequency " bottom " at the base-line.

Normally mitral valve closure precedes slightly the closure of the tricuspid valve (Leatham, 1954). This temporal separation is often exaggerated in atrial septal defect as is discussed above. Furthermore, the second component produced by tricuspid valve closure is likely to be accentuated. The result (Fig. 8) is a complex easily confused with presystolic crescendo and snapping mitral first sound.

Thoracic Arterio-Venous Fistula

Fistulæ between the aorta or its intrathoracic branches and the vena caval system or pulmonary artery produce murmurs that are continuous as a rule and have a characteristic time-frequency-intensity pattern. Murmurs that show this pattern are those of patent ductus arteriosus, the artificial ductus of the Blalock-Taussig anastomosis, aortic septal defect, intercostal or coronary

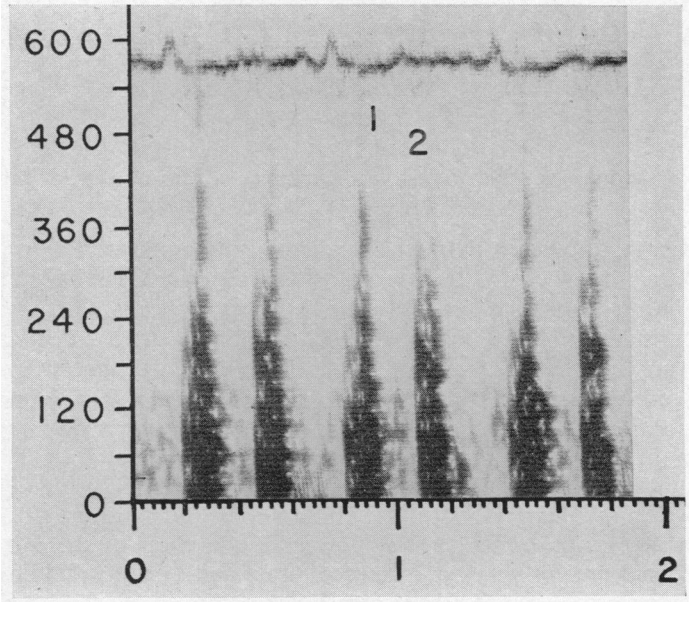


Fig. 8.—Crescendo construction of S_1 with atrial septal defect.

A-V fistulæ, and even thyroid bruits. Pulmonary A-V fistulæ probably show the same pattern. Venous hums are likewise usually indistinguishable. The principal practical usefulness is the opportunity the pattern provides for distinguishing this type of lesion from stenosis and regurgitation of the aortic or pulmonary valve. This application is well illustrated by the case represented in Fig. 13.

Fig. 9 presents the typical pattern and the features distinguishing it from that of stenosis-regurgitation at an arterial orifice. The continuous murmur of thoracic A-V fistula has a peak of intensity and frequency span in the vicinity of the second heart sound. On the other hand, in aortic stenosis and regurgitation, for example, two peaks of intensity and frequency are generally present. The ejection stenosis murmur in systole has a " Christmas tree " configuration as a result of the peak of frequency and intensity in mid-systole. The diastolic murmur has a peak at or soon after the second sound with diminuendo thereafter. In Fig. 10 is presented the typical auscultatory finding in patent ductus arteriosus.

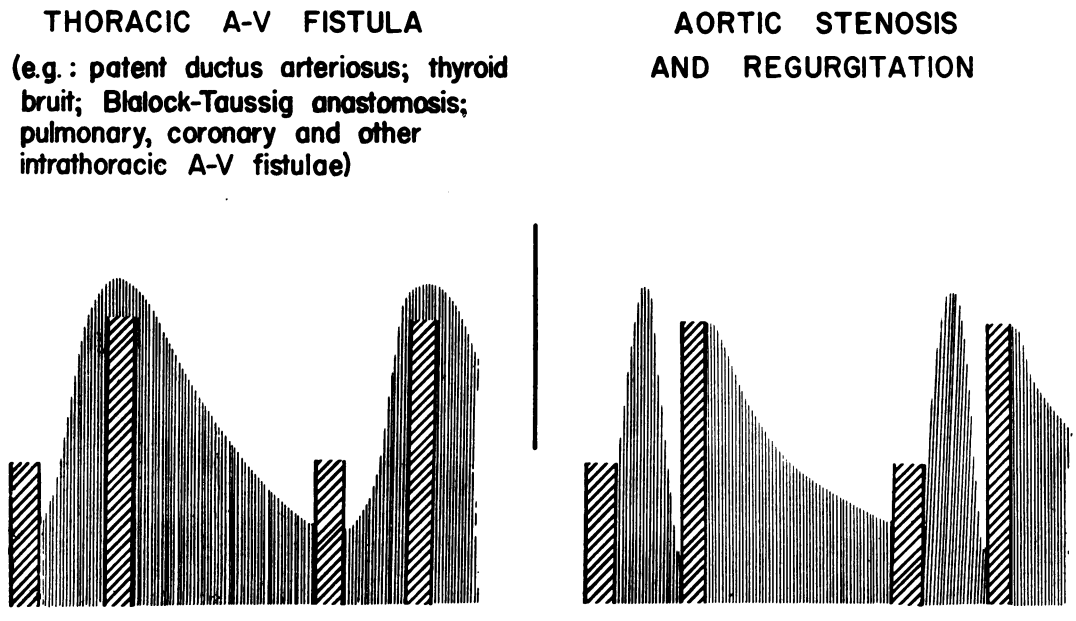


FIG. 9.—Comparison of the frequency-time pattern of the murmur of thoracic arterio-venous fistula with that of aortic stenosis and regurgitation.

As in other congenital anomalies with which a dilated pulmonary artery is associated, an early pulmonary systolic click may occur with patent ductus arteriosus. The spectral phonocardiogram of such a case has been shown elsewhere (McKusick *et al.*, 1954 *b*). Occasionally there is a pulmonary systolic murmur, produced in the dilated pulmonary artery, distinct from the systolic component of the continuous murmur. In such instances the pulmonary systolic murmur is likely to be introduced by the early systolic click. The recording in Fig. 12 is from a patient with patent ductus and pulmonary hypertension. There was no continuous murmur. In the recording from the pulmonary area there is diminuendo systolic murmur initiated by an early systolic click.

At the apex a low-pitched rumbling mid-diastolic murmur is occasionally heard with patent ductus arteriosus (Ravin and Darley, 1950). It appears to be the murmur of relative mitral stenosis resulting from two factors—torrential mitral flow and dilatation of the left ventricle. Fig. 11 presents an example in a patient with typical auscultatory and operative findings of patent ductus: in this instance the sound is circumscribed, rather suggesting a summation gallop, but it begins too late to be a third-sound gallop and too early for atrial gallop.

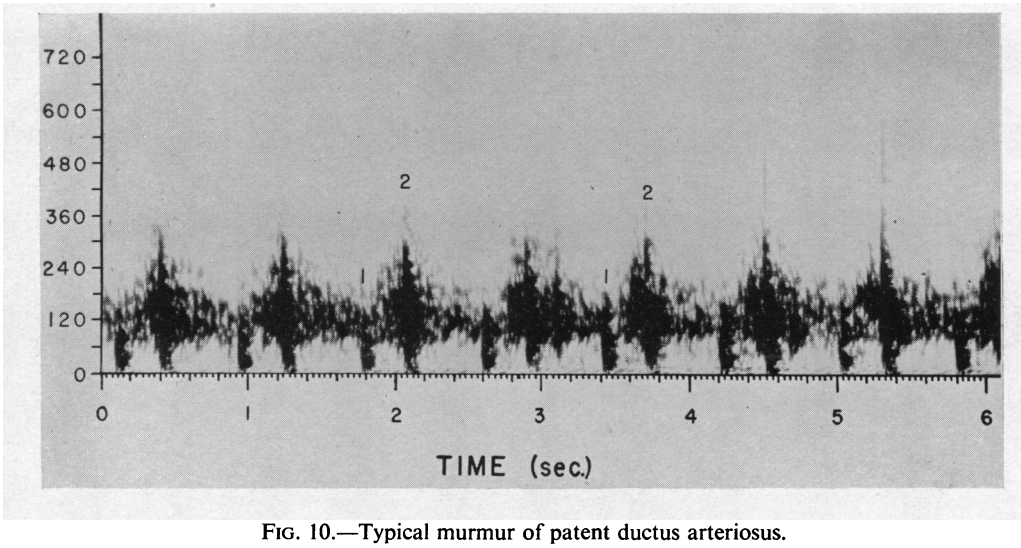


FIG. 10.—Typical murmur of patent ductus arteriosus.

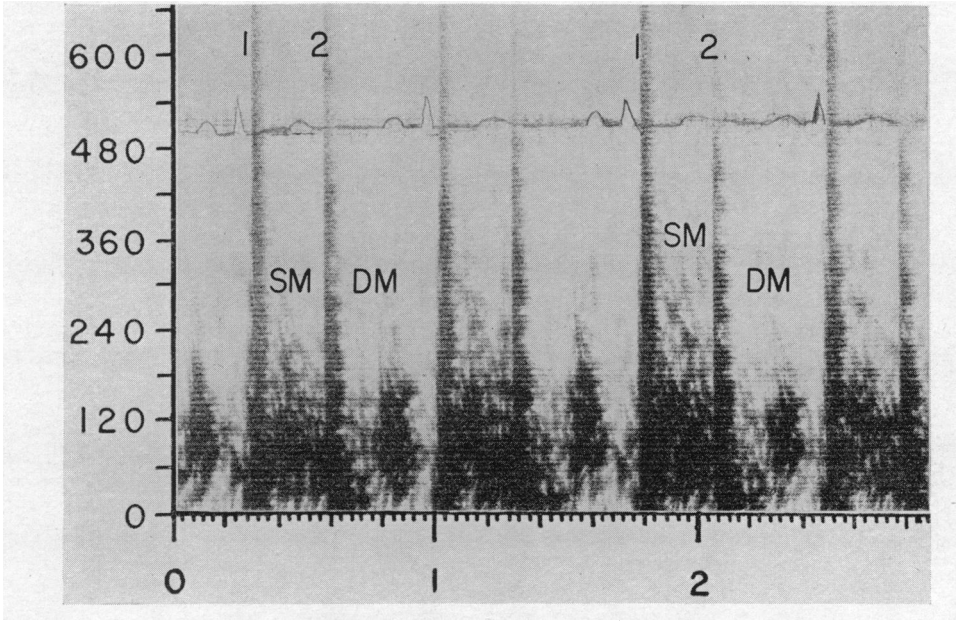


FIG. 11.—Mid-diastolic rumble at apex in patent ductus.

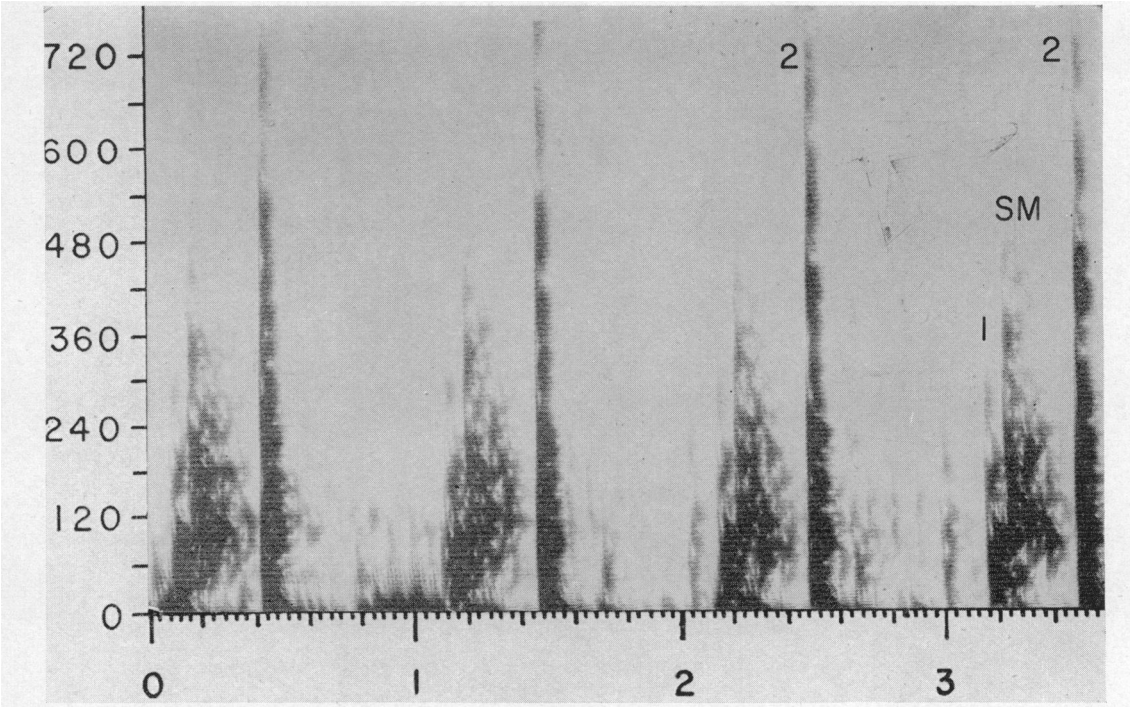


FIG. 12.—Patent ductus arteriosus with pulmonary hypertension (pulmonary area). An early systolic click initiates a diminuendo systolic murmur. P_2 is greatly accentuated.

In Fig. 13 is presented the sound recording in the third right interspace of a patient in whom this type of study was diagnostically helpful. This 50-year-old coloured woman has been followed at this hospital for 17 years. The murmur and cardiac enlargement have been present throughout but both have apparently increased slowly over the years. The patient has had virtually no symptoms referable to the heart and her pulse pressure has been normal. However, her diagnosis has been aortic stenosis and regurgitation throughout. With the passage of years right axis deviation has developed in the electrocardiogram (which previously showed normal axis) and in recent months atrial fibrillation has set in. These developments are not surprising in the light of the pronounced enlargement of the right atrium and right ventricle on angiocardiograms. The enlargement of the right side of the heart appears to be mainly a matter of dilatation with little hypertrophy. On auscultation a continuous murmur loudest in the third right interspace was heard. By stethoscope the murmur seemed to " peak " in the vicinity of the second heart sound. This impression was confirmed by the spectral phonocardiogram (Fig. 13).

Further studies in this patient have been impossible. However, the evidence of left-to-right shunt of the intrathoracic fistula type is very strong.

Bonham-Carter and Walker (1955) have recently indicated two other bases for a continuous murmur that may be difficult to distinguish from that of patent ductus arteriosus: (1) ventricular septal defect with pulmonary regurgitation; and (2) large tortuous collateral vessels as with truncus arteriosus or pulmonary atresia.

PRIMARY PULMONARY HYPERTENSION

The principal auscultatory features of two illustrative cases, one adult and one juvenile, will be demonstrated. In both the disease is thought to be of congenital origin.

The first patient, 6 years old, whose heart sounds are illustrated in Fig. 14 had a pulmonary

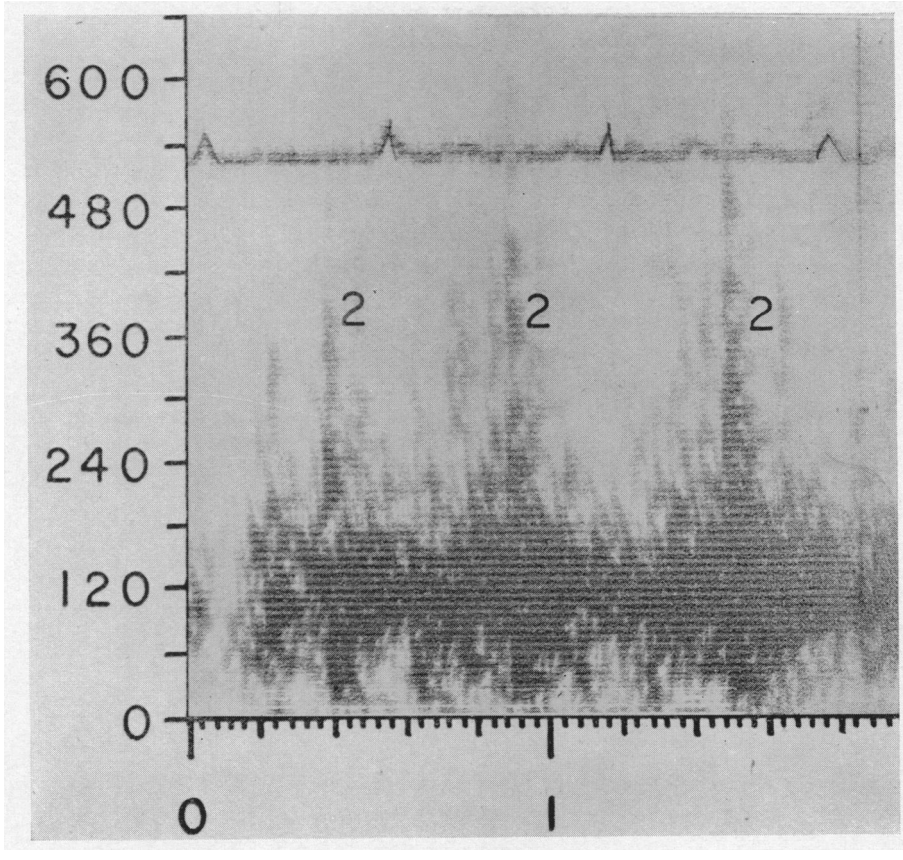


FIG. 13.—Thoracic arteriovenous fistula, congenital.

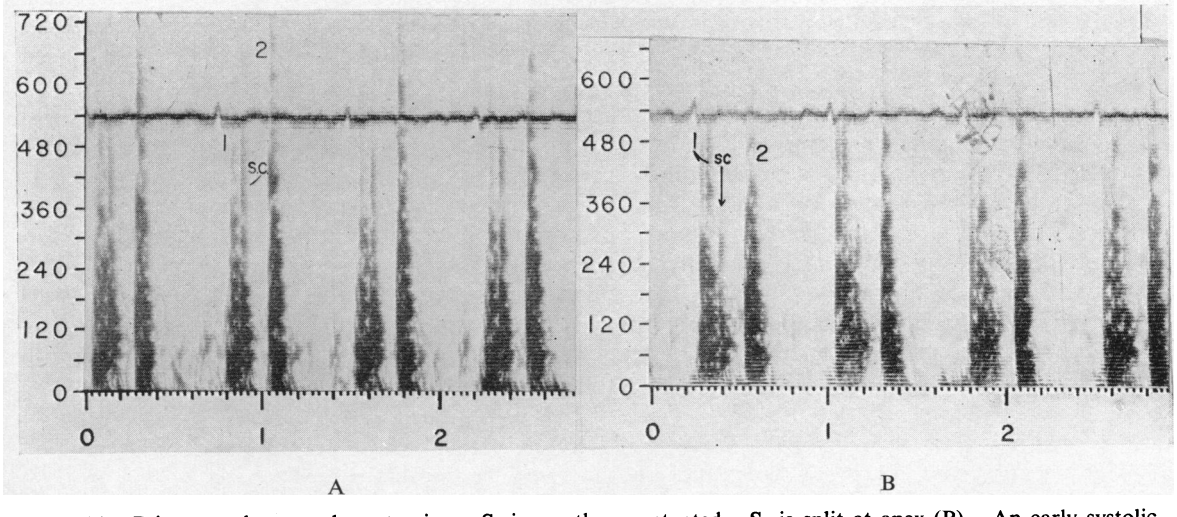


FIG. 14.—Primary pulmonary hypertension. S_2 is greatly accentuated. S_1 is split at apex (B). An early systolic click is loudest in the pulmonary area (A).

arterial pressure of 97/73 with a mean of 85 mm. Hg by cardiac catheterization and a much dilated main pulmonary artery by radiography. The features of note are as follows.

1. The second sound is greatly accentuated in both intensity and frequency span. It is not split. It is worthwhile reiterating that, contrary to the common misconception, hypertension of either arterial circuit, is not *per se* a cause of significant or consistent splitting of S_2.

2. An unusually loud early systolic click (" SC ") in the pulmonary area (Fig. 14A) understandably created a mistaken impression of widely split S_1 on auscultation. It seems to be mainly the tricuspid closure sound that is audible in the pulmonary area.

3. That the sound labelled as early systolic click is indeed so is strongly indicated by the relatively long time interval between it and QRS. The matter is clinched by examination of the recording at the apex (Fig. 14B) where three sounds are demonstrated. These can, with considerable confidence, be identified successively as mitral closure sound, tricuspid closure sound, and early systolic click.

4. An atrial gallop is present. A faint third heart sound was demonstrated in some cycles.

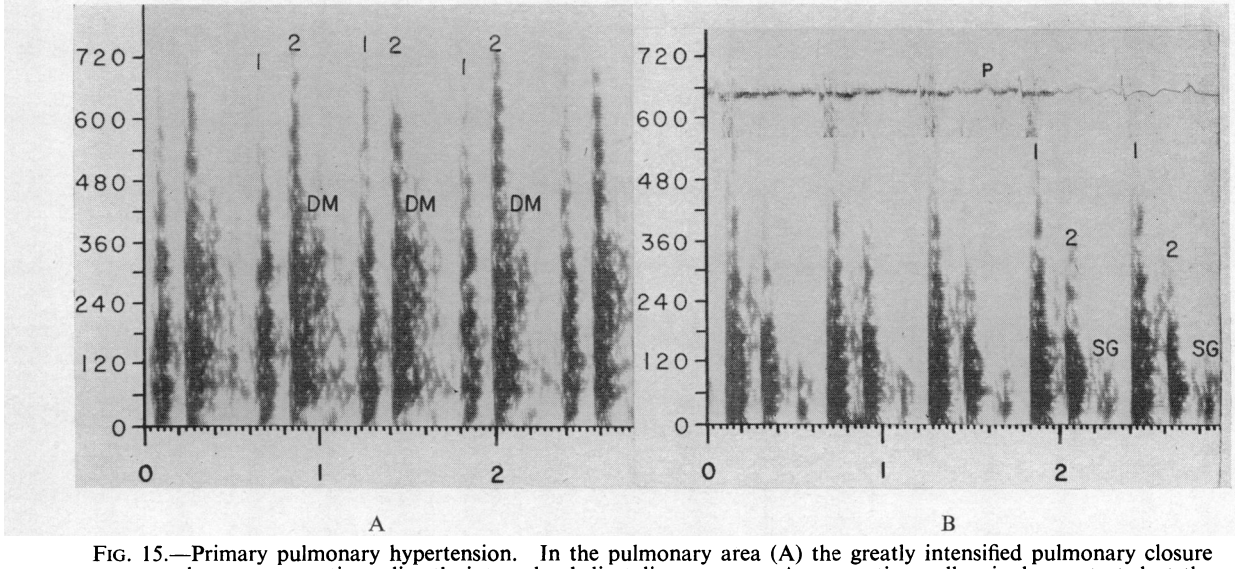


FIG. 15.—Primary pulmonary hypertension. In the pulmonary area (A) the greatly intensified pulmonary closure sound seems to continue directly into a loud diastolic murmur. A summation gallop is demonstrated at the apex (B).

The second patient, whose recordings are displayed in Fig. 15, is 29 years old. The pressure in the main pulmonary artery was 134/67 with a mean of 89 mm. Hg. At the pulmonary area (Fig. 15A) the second sound is tremendously accentuated in both intensity and in frequency span and continues directly into a diastolic murmur of the Graham-Steell type. At the apex (Fig. 15B) the first heart sound is slightly split. The second component of the split first sound, undoubtedly produced by tricuspid closure, is greatly accentuated. There is a systolic murmur. In mid-diastole there is a summation gallop. On auscultation this seemed to be a mid-diastolic rumble. However, the P–R interval was 0·26 seconds and close inspection reveals that when a longer murmur-like sound is present, two sounds, third heart sound and atrial sound, can be identified as contributing to it.

CONGENITAL MITRAL STENOSIS

Recordings were made from a one-year-old infant with classical auscultatory, radiographic (including angiocardiographic), operative, and necropsy findings of mitral stenosis. The pathological features supported the clinical opinion that the mitral valve disease was of congenital rather than rheumatic origin.

The spectral phonocardiogram in this case is identical with that seen in most cases of rheumatic mitral stenosis in adults (McKusick *et al.*, 1954 *b*; 1955). The features of note, as illustrated in Fig. 16, are as follows.

1. The mitral closure sound (1) is snapping (as indicated by the conspicuous harmonics, e.g. the one at about 420 c.p.s.), is intensified and is delayed in relation to the QRS.

2. There is a diminuendo systolic murmur denoting a certain degree of concomitant mitral regurgitation.

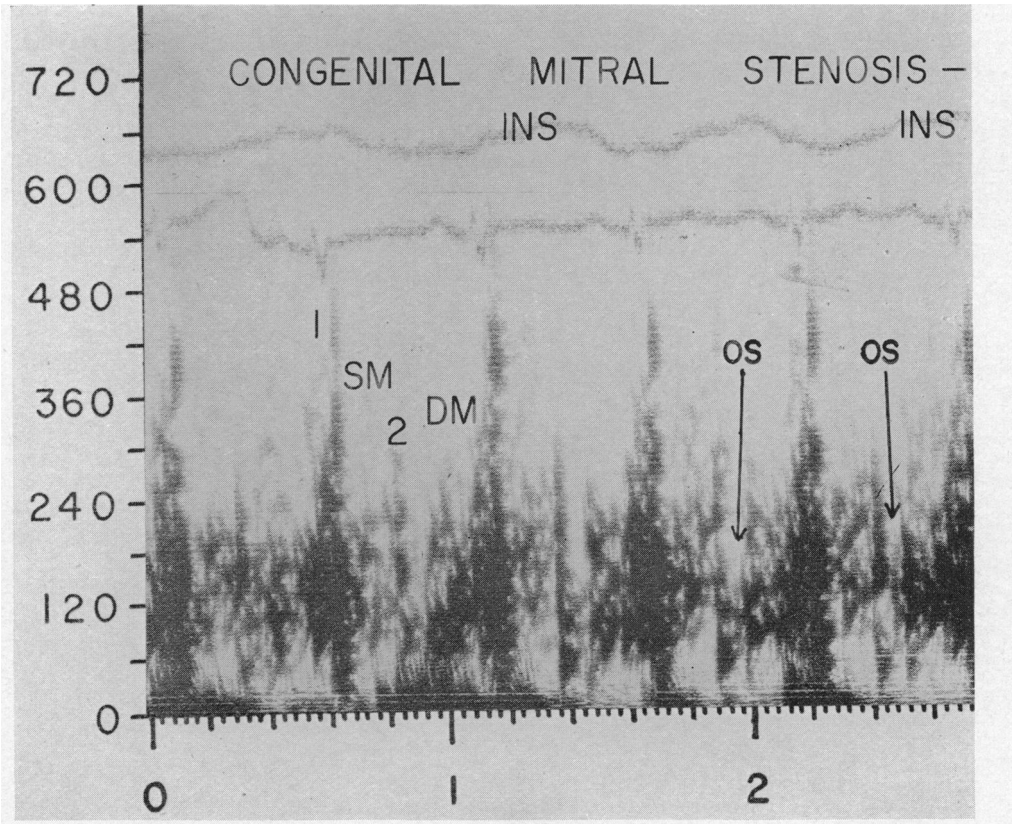


FIG. 16.—Congenital mitral stenosis. Apex. Inspiration (INS), opening snap (OS), the first and second heart sounds (1 and 2), the systolic murmur (SM), and the diastolic murmur (DM) are marked.

3. There is a suspicion of a diminutive opening snap (OS) following the second heart sound. The impression of opening snap was strengthened by examination of the recordings from the pulmonary and left lower sternal areas. These revealed, in addition to identifiable aortic and pulmonary closure sound, a sound that was located at the same point in the cardiac cycle as the presumed opening snap at the apex, which was appreciably louder at these sites and had the spectrographic characteristics previously described for snaps in general and the mitral opening snap in particular.

4. The diastolic rumble is initiated by a circumscribed sound with the temporal characteristics of a third heart sound.

5. The diastolic rumble is first diminuendo in both intensity and peak frequency.

6. Following the P wave of the electrocardiogram and building up to the snapping mitral

closure sound, the diastolic rumble becomes greatly accentuated with a crescendo in respect both to intensity and to peak frequency.

Except for the additional feature of a systolic murmur, Duroziez's onomatopœic device, " fout-ta-ta-rou," is rather accurately reproduced.

Necropsy revealed that the mitral valve was diaphragmatic with a small central perforation.

Summary and Conclusions

The spectral phonocardiographic patterns in thoracic A-V fistula (exemplified by patent ductus arteriosus), in pulmonary stenosis, and in atrial septal defect are sufficiently distinctive to suggest the diagnosis from examination of the sound recordings alone. The pulmonary reversal snap of valvular pulmonary stenosis is highly diagnostic, particularly when combined with a murmur of the typical ejection stenosis type.

Although most reported cases of congenital mitral stenosis have not had completely typical auscultatory findings in terms of the experience with rheumatic mitral stenosis, a case is presented in which typical auscultatory findings, including a mitral opening snap, did occur. Such cases might be particularly favourable for valvotomy.

Some features of the heart sounds in primary pulmonary hypertension have been demonstrated. Exaggerated splitting of the second pulmonary sound is not a feature.

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